cellular dehydration, distention of cerebral vessels, and subarachnoid and subdural hemorrhage.

Another harmful procedure is the use of acid solutions to neutralize ingested alkaline caustics. It is true that a weak acid will neutralize an alkali; however, experimental studies have shown that this reaction results in the production of heat severe enough to produce a thermal burn, further damaging the chemically burned mucosal surfaces. Milk, when used as a diluent, will decrease the caustic potential of the alkali while producing no additional heat.

The combination of activated charcoal, tannic acid and magnesium oxide, known as the "universal antidote," has been shown to be ineffective. The charcoal adsorbs the other ingredients making none of them available for their intended use. It should not be recommended or used.

ANTHONY S. MANOGUERRA, PharmD

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The Fetal Alcohol Syndrome

THE FETAL ALCOHOL SYNDROME is a pattern of altered growth, morphogenesis and function seen in the offspring of chronically alcoholic women who continue to drink heavily throughout their pregnancy. First set forth in 1973 by Jones and co-workers, the principal features of this disorder include the following: prenatal and postnatal onset growth deficiency, mental retardation with an average IQ of 63, microcephaly, short palpebral fissures, joint contractures, altered palmar crease patterns, cardiac defects and fine motor dysfunction. Approximately 40 percent of infants born to chronically alcoholic women who continue heavy alcohol consumption during pregnancy have serious problems in development. The incidence of serious developmental defects in the offspring of women who drink lesser amounts of alcohol is not known. However, recent evidence suggests that 11 percent of the offspring of women who drink one ounce of absolute alcohol a day during the first trimester of pregnancy have serious problems in development. Nothing is known about the effect of "social drinking" during the first trimester of pregnancy nor are data available on the effect of "binge drinking."

KENNETH LYONS JONES, MD

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Reye Syndrome: Evaluation and Treatment of Intracranial Pressure

INCREASED INTRACRANIAL PRESSURE (ICP) secondary to cerebral edema is the major cause of neurological deterioration in patients with Reye syndrome. Increased intracranial pressure should be evaluated early in the course of treatment before irreversible brain dysfunction has occurred. In the presence of pronounced ICP, a lumbar puncture can be hazardous, risking brain-stem herniation. However, when the possibility of meningitis exists, a spinal tap is indicated. Pretreatment with corticosteroids and mannitol may substantially reduce the risk of herniation in these patients. Computerized axial tomography is extremely useful in showing cerebral edema with visualization of diffuse brain swelling and decreased ventricular size, and can usually show if a subdural hematoma is present.

Although independent evaluations should be done in each patient, routine protocol staging will be useful in guiding treatment and in prognosis. Careful monitoring of fluid given intravenously and of osmotic agents such as mannitol (1.5 to 3 grams per kg of body weight given intravenously), urea (1 to 1.5 grams per kg of body weight given intravenously) or glycerol (1.5 grams per kg of body weight given orally via nasogastric tube), and dexamethasone (1 mg per year of age given intravenously every four hours) can be useful in controlling ICP. However, the single most important aspect of reducing ICP is to insure adequate ventilation. Frequent arterial blood gas determinations should be obtained because respiratory intervention may be necessary. Mild hypothermia also may be useful in reducing ICP.

Severe ICP can be monitored with either an epidural or a direct ventricular catheter attached to a pressure transducer. Control of increased intracranial pressure then can be effected using a combination of mannitol and dexamethasone.